DETAILED ACTION

1. Applicant's response filed 3/23/11 is acknowledged and has been entered.

2. Applicant' is reminded of Applicant's election without traverse of the Invention of Group I and the species of isolated antibody DF200, a detectable moiety, IL-2 as the additional component, and antibody that binds to KIR2DL1 and KIR2DL2/3 and neutralizes KIR mediated inhibition of NK cell cytotoxicity, in Applicant's amendment filed 4/24/09

Claims 70-79, 81 and 83-91 are presently being examined.

3. Applicant's amendment filed 12/2/10 has overcome the prior rejection of record of claim 80 under 35 U.S.C. 103(a) as being obvious over US 2005/0037002 A1 (of record) in view of Eisenthal *et al* (J. of Immunol. 1990, 144: 4463-4471).

4. Applicant's response filed 3/23/11 and evidence filed on the same date has overcome the prior rejection of record of claims 88 and 90-91 are rejected under 35 U.S.C. 102(b) as being anticipated by Kim *et al* (J. Immunol. 1997, 159: 3875-3882) as evidenced by Shin *et al* (Hybridoma, 1999, 18(6): 521-527) and by admissions in the specification on page 25 at lines 15-28 and at the paragraph spanning pages 25-26.

5. Applicant's response filed 3/23/11 and evidence filed on the same date has overcome the prior rejection of record of claims 88 and 89 under 35 USC 103(a) as being obvious over Kim *et al* (J. Immunol. 1997, 159: 3875-3882) in view of Harlow and Lane.

6. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

7. Claims 88 and 90 are rejected under 35 U.S.C. 102(b) as being anticipated by Spaggiari *et al* (Blood, 2002, 99(5): 1076-1714, IDS reference) as evidenced by AbD Serotec and as evidenced by an admission in the specification on page 62 at lines 4-5 and 9.

Spaggiari *et al* teach the NKVFS1 mAb that recognizes a common epitope of CD158a (KIR2DL1) and CD158b (KIR2DL2/3) (especially materials and methods section on page 1706).

Evidentiary reference AbD Serotec teaches that NKVFS1 specifically inhibits NK cell cytotoxicity of the KIR2DL forms.

With regard to the limitation recited in instant claim 90 "wherein said antibody or fragment thereof competes for binding to said KIR2DL1 and/or KIR2DL2/3 on the surface of an NK cell with antibody DF200", the admission in the specification on page 62 at lines 4-5 and 9 is that NKVSF1 competes with DF200 for binding to KIR2DL forms. Therefore the claimed antibody appears to be the same as the antibody of the prior art absent a showing of differences. Since the Patent Office does not have the facilities for examining and comparing the mAb of the instant invention to those of the prior art, the burden is on Applicant to show a distinction between the antibody of the instant invention and that of the prior art. See In re Best, 562 F.2d 1252, 195 USPQ 430 (CCPA 1977).

8. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

9. Claims 88, 90 and 91 are rejected 35 U.S.C. 103(a) as being obvious over Shin *et al* (Hybridoma, 1999, 18(6): 521-527) in view of Kim *et al* (J. Immunol. 1997, 159: 3875-3882, of record) as evidenced by admissions in the specification on page 25 at lines 15-28 and at the paragraph spanning pages 25-26.

Shin *et al* teach that HLA-C-recognizing receptor that is inhibitory is the p58 belonging to KIR2DL group comprised of KIR2DL1 and KIR2DL2/3 (especially paragraph spanning pages 521-522). Shin *et al* teach that the mAb that has its epitope in the HLA-binding region in p58 KIR may be the most effective mAb for blocking the interaction between p58 KIR and HLA-C that it is known that both the γ2 and γ3 domains are involved in the interaction between p58 KIR and its ligand, HLA-C (especially second full paragraph at line 1, column 1 on page 526). Shin *et al* teach the method of mAb production via conventional hybridoma technology of Kohler and Milstein as well as methods for assessing the ability of the mAb to inhibit p58-mediated inhibition of NK cell cytotoxicity (especially materials and methods section).

Shin *et al* do not exemplify wherein the anti-p58 mAb blocks the binding between p58 KIR and HLA-C, nor that it competes for binding with mAb DF200 to KIR2DL1 and/or KIR2DL2/3.

Kim *et al* teach that a polypeptide consisting of the two extracellular Ig domains can be recombinantly produced, as it folds properly. Kim *et al* teach that their experiments suggest that both Ig domains of p58 are necessary for HLA-C binding and that the binding site on KIR might be the exposed region at the interface between the N- and C-terminal γ domains (see entire reference, especially paragraph spanning columns 1-2 on page 3879). Kim *et al* teach anti-p58 KIR mAbs that were found to interefere with

class I-mediated protection of target cells, *i.e.*, they are capable of neutralizing KIR-mediated inhibition of NK cell cytotoxicity in NK cells expressing p58 (especially page 3876).

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have made more mAbs by the conventional hybridoma monoclonal antibody methodology taught by Shin *et al*, including to have used the γ2 and γ 3 domain peptide as an immunogen, to have tested and selected for antibodies that bind to both KIR2DL1 and to KIR2DL2/3 and to have further tested these antibodies for the ability to neutralize KIR-mediated inhibition of NK cell cytotoxicity.

With regard to the limitation recited in instant claim 90, that the mAb competes for binding to KIR2DL1 and/or KIR2DL2.3 on the surface of an NK cell with antibody DF200 produced by the hybridoma deposited as CNCM I-3224, although the art references do not explicitly teach said ability to compete, the mAb taught by the combined references interferes with the class I mediated protection of target cells, *i.e.,* that it interferes with the interaction of HLA-C with p58 and neutralizes p58-mediated NK cell cytotoxicity. Like-wise, the antibody DF200 produced by hybridoma CNCM I-3224, possesses this same functional activity (see the recitation in claim 70), while the primary art reference teaches that a neutralizing antibody recognizes an epitope in the HLA-binding region of p58. Also note that the definition in the specification of "neutralize KIR-mediated inhibition of NK cell cytotoxicity" may be partial or full neutralization (see below***).

Therefore the claimed antibody appears to be the similar to the antibody of the prior art absent a showing of unobvious differences. Since the Patent Office does not have the facilities for examining and comparing the mAb of the instant invention to those of the prior art, the burden is on Applicant to show an unobvious distinction between the antibody of the instant invention and that of the prior art. See In re Best, 562 F.2d 1252, 195 USPQ 430 (CCPA 1977).

***The admissions in the instant specification on page 25 at lines 15-28 is: "Antibodies of this invention may partially or fully neutralize the KIR-mediated inhibition of NK cell cytotoxicity. The term "neutralize KIR-mediated inhibition of NK cell cytotoxicity," as used herein means the ability to increase to at least about 20%, preferably to at least about 30%, at least about 40%, at least about 50% or more (e.g., about 25-100%) of specific lysis obtained at the same ratio with NK cells or NK cell lines that are not blocked by their KIR, as measured by a classical chromium release test of cytotoxicity, compared with the level of specific lysis obtained without antibody when an NK cell population expressing a given KIR is put in contact with a target cell expressing the cognate MHC class I molecule (recognized by the KIR expressed on NK cell). For example, preferred antibodies of this invention are able to induce the lysis of matched or HLA compatible or autologous target cell populations, i.e., cell populations that would not be effectively lysed by NK cells in the absence of said antibody. Accordingly, the antibodies of this invention may also be defined as facilitating NK cell activity in vivo."

The admissions in the instant specification at the paragraph spanning pages 25-26 is: "Alternatively, the term "neutralize KIR mediated inhibition" means that in a chromium assay using an NK cell clone or transfectant expressing one or several inhibitory KIRs and a target cell expressing only one HLA allele that is recognized by one of the KIRs on the NK cell, the level of cytotoxicity obtained with the antibody

should be at least about 20%, preferably at least about 30%, at least about 40%, at least about 50% (e.g., about 25-100%), or more of the cytotoxicity obtained with a known blocking anti MHC class I molecule, such as W6/32 anti MHC class I antibody."

Applicant's arguments have been fully considered but are not persuasive.

Applicant's arguments are of record in the response filed 3/23/11 on pages 6-8, in brief, that there is no motivation to seek antibodies that bind to both KIR2DL1 and KIRDL2/3 and neutralize KIR-mediated inhibition of cytotoxicity, one of ordinary skill in the art would not have had a reasonable expectation of success in making antibodies that bound to both KIR2DL1 and KIR2DL2/3 and neutralized KIR-mediated inhibition of NK cell cytotoxicity. Applicant asserts that there is no indication that Shin do anything other than simply consider KIR2DL1 and KIR2DL2/3 separately, given that they bind to different HLA-Cw receptors (cw-4 and cw2-3, respectively). Applicant asserts that while the two share high homology, the homology is lower in the HLA binding regions and involve different amino acid residues. Applicant argues that this would have directed a person of skill in the art away from the idea of an antibody that bound both KIR in a mutually exclusive fashion and that was able to neutralize KIR-mediated inhibition of NK cell cytotoxicity.

However, the antibodies of Shin *et al* were produced using standard hybridoma technology, and Applicant's working examples 2-7 also use standard hybridoma technology in order to produce antibodies that recognize both aforementioned KIR2DL receptors and neutralize KIR-mediated inhibition of NK cell cytotoxicity. Applicant makes assertions concerning the degree of homology between the two KIR2DL receptors (in fact, a single reciprocal amino acid residue interchange between the KIR2DL members can switch the binding of the HLA-Cw molecule to its respective KIR2DL receptor to the specificity of the other member receptor; see below at item #12 of this Office Action), but does not present evidence of nonobviousness. Applicant appears to be arguing it was not obvious to produce the claimed antibodies, but Applicant pursued their production anyway using same standard hybridoma technology. With regard to the allegation of teaching away from the claimed invention because the teaching in Shin *et al* is that the mAb should have its epitope in the HLA binding region in p58 KIR2DL forms, Applicant appears to be arguing that antibody binding to the ligand KIR2DL receptor is equivalent to KIR2DL receptors binding to their respective HLA molecules. This argument is not persuasive, as antibodies bind their ligands differently than the KIR2DL receptors bind to their respective HLA molecules. It was well known to one of ordinary skill in the art at the time of invention that antibodies may bind conformational as well as linear determinants.

10. Claims 88-91 are rejected 35 U.S.C. 103(a) as being obvious over Shin *et al* (Hybridoma, 1999, 18(6): 521-527 in view of Kim *et al* (J. Immunol. 1997, 159: 3875-3882, of record) as evidenced by admissions in the specification on page 25 at lines 15-28 and at the paragraph spanning pages 25-26, and further in view of Harlow and Lane (of record).

The combination of Shin *et al* in view of Kim *et al* as evidenced by the cited admissions in the specification, has been discussed supra.

The said combination does not teach wherein the antibody is comprised in a composition with a pharmaceutically acceptable excipient.

Harlow and Lane teach that PBS or similar isotonic solutions are commonly used buffers for storing purified antibodies (page 287 at item 1).

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have placed the antibodies taught by Shin *et al* in PBS as taught by Harlow and Lane.

One of ordinary skill in the art would have been motivated to do this in order to store the anti-p58 antibodies.

Applicant's arguments have been fully considered but are not persuasive.

Applicant's arguments are of record in the response filed 3/23/11 on page 8.

The Examiner's response to Applicant's arguments presented supra also apply herein.

11. Claims 88-91 are rejected 35 U.S.C. 103(a) as being obvious over Spaggiari *et al* (Blood, 2002, 99(5): 1076-1714, IDS reference) in view of Harlow and Lane (of record), as evidenced by AbD Serotec and as evidenced by an admission in the specification on page 62 at lines 4-5 and 9.

Spaggiari *et al* teach the NKVFS1 mAb that recognizes a common epitope of CD158a (KIR2DL1) and CD158b (KIR2DL2/3) (especially materials and methods section on page 1706).

Spaggiari *et al* do not teach wherein the mAb is present in a composition with a pharmaceutically acceptable excipient.

A pharmaceutically acceptable carrier such as PBS was well known in the art as solvent for immunoglobulins for storage and immunoassays as taught by Harlow (see page 287, Harlow, 1988).

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have placed the mAb taught by Spaggiari *et al* in PBS.

One of ordinary skill in the art at the time the invention was made would have been motivated to do this in order to store the mAb.

Evidentiary reference AbD Serotec teaches that NKVFS1 specifically inhibits NK cell cytotoxicity of the KIR2DL forms.

With regard to the limitation recited in instant claim 90 "wherein said antibody or fragment thereof competes for binding to said KIR2DL1 and/or KIR2DL2/3 on the surface of an NK cell with antibody DF200", the admission in the specification on page 62 at lines 4-5 and 9 is that NKVSF1 competes with DF200 for binding to KIR2DLforms. Therefore the claimed antibody appears to be similar to the antibody of the prior art absent a showing of unobvious differences. Since the Patent Office does not have the facilities for examining and comparing the mAb of the instant invention to those of the prior art, the burden is on Applicant to show an unobvious distinction between the antibody of the instant invention and that of the prior art. See In re Best, 562 F.2d 1252, 195 USPQ 430 (CCPA 1977).

12. Claims 88, 90 and 91 are rejected 35 U.S.C. 103(a) as being obvious over Shin *et al* (Hybridoma, 1999, 18(6): 521-527 in view of Kim *et al* (J. Immunol. 1997, 159: 3875-3882, of record) and Winter and Long (J. Immunol. 1997, 158: 4026-4028) as evidenced by admissions in the specification on page 25 at lines 15-28 and at the paragraph spanning pages 25-26.

Shin *et al* teach that HLA-C-recognizing receptor that is inhibitory is the p58 belonging to KIR2DL group comprised of KIR2DL1 and KIR2DL2/3 (especially paragraph spanning pages 521-522). Shin *et al* teach that the mAb that has its epitope in the HLA-binding region in p58 KIR may be the most effective mAb for blocking the interaction between p58 KIR and HLA-C that it is known that both the γ2 and γ3 domains are involved in the interaction between p58 KIR and its ligand, HLA-C (especially second full paragraph at line 1, column 1 on page 526). Shin *et al* teach the method of mAb production via conventional hybridoma technology of Kohler and Milstein as well as methods for assessing the ability of the mAb to inhibit p58-mediated inhibition of NK cell cytotoxicity (especially materials and methods section).

Shin *et al* do not exemplify wherein the anti-p58 mAb blocks the binding between p58 KIR and HLA-C, nor that it competes for binding with mAb DF200 to KIR2DL1 and/or KIR2DL2/3.

Kim *et al* teach that a polypeptide consisting of the two extracellular Ig domains can be recombinantly produced, as it folds properly. Kim *et al* teach that their experiments suggest that both Ig domains of p58 are necessary for HLA-C binding and that the

binding site on KIR might be the exposed region at the interface between the N- and C-terminal γ domains (see entire reference, especially paragraph spanning columns 1-2 on page 3879). Kim *et al* teach anti-p58 KIR mAbs that were found to interefere with class I-mediated protection of target cells, *i.e.*, they are capable of neutralizing KIR-mediated inhibition of NK cell cytotoxicity in NK cells expressing p58 (especially page 3876).

Winter and Long teach that changing a single amino acid residue between the p58 KIR (*i.e.*, KIR2DLA and KIR2DL2/3) correlated with a switch in the specificity of each from HLA-Cw*0401 to HLA-C2*0304, and *vice versa*. Winter and Long teach that this amino acid position is in the first Ig domain of these KIR that determines their ability to discriminate between the two groups of HLA-C allotypes. Winter and Long also teach that the two p58 receptors differ by only 17 amino acid residues in their extracellular region, with five of eleven amino acid differences upstream of the second Ig domain potentially accounting for the specificity of KIR binding to the HLA-Cw3 *vs* HLA-CW4 cluster (especially abstract, introduction, Figures 1 and 3, results and discussion at the first paragraph).

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have made more mAbs by the conventional hybridoma monoclonal antibody methodology taught by Shin *et al*, including using the γ2 and γ3 domains as an immunogen, to have tested and selected for antibodies that bind to both KIR2DL1 and to KIR2DL2/3 and to have further tested these antibodies for the ability to neutralize KIR-mediated inhibition of NK cell cytotoxicity.

With regard to the limitation recited in instant claim 90, that the mAb competes for binding to KIR2DL1 and/or KIR2DL2.3 on the surface of an NK cell with antibody DF200 produced by the hybridoma deposited as CNCM I-3224, although the art reference does not explicitly teach said ability to compete, the mAb taught by the combined references interferes with the class I mediated protection of target cells, *i.e.*, that it interferes with the interaction of HLA-C with p58 and neutralizes p58-mediated NK cell cytotoxicity. Like-wise, the antibody DF200 produced by hybridoma CNCM I-3224, possesses this same functional activity (see the recitation in claim 70), while the primary art reference teaches that a neutralizing antibody recognizes an epitope in the HLA-binding region of p58. Also note that the definition in the specification of "neutralize KIR-mediated inhibition of NK cell cytotoxicity" may be partial or full neutralization (see below***).

Therefore the claimed antibody appears to be the similar to the antibody of the prior art absent a showing of unobvious differences. Since the Patent Office does not have the facilities for examining and comparing the mAb of the instant invention to those of the prior art, the burden is on Applicant to show an unobvious distinction between the antibody of the instant invention and that of the prior art. See In re Best, 562 F.2d 1252, 195 USPQ 430 (CCPA 1977).

***The admissions in the instant specification on page 25 at lines 15-28 is: "Antibodies of this invention may partially or fully neutralize the KIR-mediated inhibition of NK cell cytotoxicity. The term "neutralize KIR-mediated inhibition of NK cell cytotoxicity," as used herein means the ability to increase to at least about 20%, preferably to at least about 30%, at least about 40%, at least about 50% or more (e.g., about 25-100%) of specific lysis obtained at the same ratio with NK cells or NK cell lines that are not blocked by their KIR, as measured by a classical chromium release test of cytotoxicity, compared with the level of specific lysis obtained without antibody when an NK cell population expressing a given KIR is put in contact with a target cell expressing the cognate MHC class I molecule (recognized by the KIR expressed on NK cell). For example, preferred antibodies of this invention are able to induce the lysis of matched or HLA compatible or autologous target cell populations, i.e., cell populations that would not be effectively lysed by NK cells in the absence of said antibody. Accordingly, the antibodies of this invention may also be defined as facilitating NK cell activity in vivo."

The admissions in the instant specification at the paragraph spanning pages 25-26 is : "Alternatively, the term "neutralize KIR mediated inhibition" means that in a chromium assay using an NK cell clone or transfectant expressing one or several inhibitory KIRs and a target cell expressing only one HLA allele that is recognized by one of the KIRs on the NK cell, the level of cytotoxicity obtained with the antibody should be at least about 20%, preferably at least about 30%, at least about 40%, at least about 50% (e.g., about 25-100%), or more of the cytotoxicity obtained with a known blocking anti MHC class I molecule, such as W6/32 anti MHC class I antibody."

13. Claims 88-91 are rejected 35 U.S.C. 103(a) as being obvious over Shin *et al* (Hybridoma, 1999, 18(6): 521-527 in view of Kim *et al* (J. Immunol. 1997, 159: 3875-3882, of record) and Winter and Long (J. Immunol. 1997, 158: 4026-4028) as evidenced by admissions in the specification on page 25 at lines 15-28 and at the paragraph spanning pages 25-26, and further in view of Harlow and Lane (of record).

The combination of Shin *et al* in view of Kim *et al* and Winter and Long as evidenced by the cited admissions in the specification, has been discussed supra.

The said combination does not teach wherein the antibody is comprised in a composition with a pharmaceutically acceptable excipient.

Harlow and Lane teach that PBS or similar isotonic solutions are commonly used buffers for storing purified antibodies (page 287 at item 1).

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have placed the antibodies taught by the combined references in PBS as taught by Harlow and Lane.

One of ordinary skill in the art would have been motivated to do this in order to store the anti-p58 antibodies.

Applicant's arguments have been fully considered but are not persuasive.

Applicant's arguments are of record in the response filed 3/23/11 on page 8.

The Examiner's response to Applicant's arguments presented supra also apply herein.

14. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.
Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

15. Claims 70-79, 81 and 83-91 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 5-7 of copending Application No. 12,813,040. Although the conflicting claims are not identical, they are not patentably distinct from each other because the DF-200 antibody is a common and obvious variant of the antibody, fragment or derivative of that in the claims of '040 that comprise one or more light variable region CDRs, the light chain variable region sequence, one or more of the heavy chain variable region CDRs or the heavy chain variable region sequence of DF-200 recited in the claims of '040. In addition, Fab or F(ab')2 and chimeric or humanized antibodies are common and obvious variants of antibodies. In addition, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have bound the antibody or fragment thereof of the claims of '040 to a solid support or a detectable moiety or to have placed it in a pharmaceutically acceptable excipient such as PBS. One of ordinary skill in the art at the time the invention was made would have been motivated to do this in order to use the antibody in a detection assay.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

16. Claims 88-91 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 3 and 4 copending Application No. 12,813,040. Although the conflicting claims are not identical, they are not patentably distinct from each other because the Pan2D antibody (also known as NKVSF1) is a common and obvious variant of the antibody or fragment or derivative of claims 3 and 4 of '040 that comprises one or more light variable region CDRs of Pan2D or that comprises the light chain variable region sequence of Pan 2D, respectively. NKVSF1/Pan2D antibody has the functional properties recited in instant claims 88 and 90. (See the art rejections of record supra). In addition, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have placed the antibody recited in claims 3 and 4 of '040 in a pharmaceutically acceptable excipient such as PBS. One of ordinary skill in the art at the time the invention was made would have been motivated to do this in order to use the antibody in a detection assay.

17. Claims 70-79, 81 and 83-91 are directed to an invention not patentably distinct from claims 5-7 of commonly assigned 12,813,040 as enunciated supra.

18. Claims 88-91 are directed to an invention not patentably distinct from claims 3 and 4 of commonly assigned 12,813,040 as enunciated supra.

19. The U.S. Patent and Trademark Office normally will not institute an interference between applications or a patent and an application of common ownership (see MPEP Chapter 2300). Commonly assigned 12,813,040, discussed above, would form the basis for a rejection of the noted claims under 35 U.S.C. 103(a) if the commonly assigned case qualifies as prior art under 35 U.S.C. 102(e), (f) or (g) and the conflicting inventions were not commonly owned at the time the invention in this application was made. In order for the examiner to resolve this issue, the assignee can, under 35 U.S.C. 103(c) and 37 CFR 1.78(c), either show that the conflicting inventions were commonly owned at the time the invention in this application was made, or name the prior inventor of the conflicting subject matter.

A showing that the inventions were commonly owned at the time the invention in this application was made will preclude a rejection under 35 U.S.C. 103(a) based upon the commonly assigned case as a reference under 35 U.S.C. 102(f) or (g), or 35 U.S.C. 102(e) for applications pending on or after December 10, 2004.

20. No claim is allowed.

21. Any inquiry concerning this communication or earlier communications from the Examiner should be directed to Marianne DiBrino whose telephone number is 571-272-0842. The Examiner can normally be reached on Monday, Tuesday, Thursday and Friday.

If attempts to reach the Examiner by telephone are unsuccessful, the Examiner's supervisor, Phuong "Neon" Huynh, can be reached on 571-272-0846. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

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